# Glycemic Index and Serum High-Density Lipoprotein Cholesterol Concentration Among US Adults

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**Background:** Dietary glycemic index, an indicator of the ability of the carbohydrate to raise blood glucose levels, and glycemic load, the product of glycemic index and carbohydrate intake, have been positively related to risk of coronary heart disease. However, the relationships between glycemic index and glycemic load and high-density lipoprotein cholesterol (HDL-C) concentration in the US population are unknown.

**Methods:** Using data from 13907 participants aged 20 years and older in the Third National Health and Nutrition Examination Survey (1988-1994), we examined the relationships between glycemic index and glycemic load, which were determined from a food frequency questionnaire and HDL-C concentration.

**Results:** The age-adjusted mean HDL-C concentrations for increasing quintiles of glycemic index distribution were 1.38, 1.32, 1.30, 1.26, and 1.27 mmol/L ($P<.001$ for trend). (To convert millimoles per liter to milligrams per deciliter, divide by 0.0259.) After additional adjustment for sex, ethnicity, education, smoking status, body mass index, alcohol intake, physical activity, energy fraction from carbohydrates and fat, and total energy intake, the mean HDL-C concentrations for ascending quintiles of glycemic index were 1.36, 1.31, 1.30, 1.27, and 1.28 mmol/L ($P<.001$ for trend). Adjusting for the same covariates and considering glycemic index as a continuous variable, we found a change in HDL-C concentration of −0.06 mmol/L per 15-unit increase in glycemic index ($P<.001$). The multiple $R^2$ for the model was 0.23. Similarly, the multivariate-adjusted mean HDL-C concentrations for ascending quintiles of glycemic load distribution were 1.35, 1.31, 1.31, 1.30, and 1.26 mmol/L ($P<.001$ for linear trend). The inverse relationships between glycemic index and glycemic load and HDL-C persisted across all subgroups of participants categorized by sex or body mass index.

**Conclusions:** These findings from a nationally representative sample of US adults suggest that high dietary glycemic index and high glycemic load are associated with a lower concentration of plasma HDL-C.

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CARDIOVASCULAR disease remains the leading cause of mortality in the United States, despite notable declines in the cardiovascular-related mortality rate since the 1960s.[1] High-density lipoprotein cholesterol (HDL-C) is a powerful predictor of the development of coronary heart disease.[2-4] Various factors are associated with HDL-C concentrations, including sociodemographic characteristics, physical activity, body mass index (BMI), alcohol use, cigarette smoking, and diet.[2] In metabolic studies, Katan[5] showed that increased intake of carbohydrates reduces HDL-C concentrations. Besides the quantitative relationship between carbohydrate intake and HDL-C concentrations, the quality of the carbohydrate characterized by glycemic index may also affect HDL-C concentrations. For example, a recent study[6] reported that glycemic index was the only dietary variable that was associated with HDL-C concentrations in a national study of the adult population in England.

The glycemic index of foods reflects their tendency to affect postprandial glucose and insulin concentrations.[7] Thus, given equal amounts of carbohydrate, food with a high glycemic index leads to higher postprandial glucose and insulin concentrations than food with a low glycemic index. Although it has been long known that foods affect glucose and insulin concentrations differently, the clinical significance of the glycemic index remains controversial.[8] Current dietary guidelines in the United States do not recommend the use of glycemic index, although many recommendations are generally consistent with the consumption of foods with a low glycemic index and avoidance of refined foods with a high glycemic index.[9,10] Examples of foods with a lower glycemic index include various legumes, pasta, and minimally refined prod-

## PARTICIPANTS AND METHODS

Started in 1988 and completed in 1994, NHANES III included a representative sample of the noninstitutionalized civilian population, which was selected by using a multistage, stratified sampling design. Persons 60 years and older and African American and Mexican American persons were oversampled. After a home interview, participants were invited to attend 1 of 3 examination sessions: morning, afternoon, or evening. For some participants who were unable to attend the examination because of health reasons, a blood sample was obtained during the home interview. Persons who attended the morning session were asked to fast for 12 hours before the session. Those who attended the afternoon or evening session were asked to fast for 6 hours.

Serum HDL-C was measured enzymatically (Hitachi 704 Analyzer; Boehringer Mannheim Diagnostics, Indianapolis, Ind) after precipitation of other lipoproteins with a manganese chloride–heparin solution. Details about quality-control procedures have been published elsewhere.[16] Values are reported in millimoles per liter; to convert to milligrams per deciliter, divide by 0.0259.

Dietary variables were created from responses to a food frequency questionnaire administered to participants to assess their usual diet over the past month. Respondents were asked how often over the past month they had eaten specified food items. If the frequency of consumption was reported as never, the value was recorded as zero. Based on previous work,[11,14] we determined glycemic index values for foods reported on the food frequency questionnaire. For example, glycemic index values were 102% for potato, 100% for white bread, 55% for apple, and 13% for broccoli. In particular, using the method described by Liu,[17,18] we calculated dietary glycemic load by multiplying the carbohydrate content of each food by its glycemic index value; this value was then multiplied by the frequency of consumption and summed for all food items.[11] Values for the carbohydrate content of each food were obtained from the US Department of Agriculture food composition tables.[19] We calculated the average glycemic index for each participant by dividing each person's glycemic load scores by their total daily intake of carbohydrates. Because individual food portion size was not collected in NHANES III, we applied standard serving sizes according to values published in the US Department of Agriculture food composition tables.[19] Previous studies[20-22] showed that additional queries on portion size for participants provided few changes in participants' ranking according to their relative intake of foods and nutrients.

We included other variables in the analyses: age, sex, race or ethnicity (white, African American, Mexican American, or other), education (years of attendance), smoking status (current, former, or never), BMI (calculated as weight in kilograms divided by the square of height in meters), alcohol consumption (drinks per month), leisure-time physical activity, energy fraction from protein and carbohydrates, and total energy intake. Alcohol consumption was determined from responses to a food frequency questionnaire. For regression models, we created a physical activity index by summing the products of the frequency of participation in 1 of 9 specific activities or up to 4 additional self-reported activities by the metabolic equivalent level for each reported activity. The energy fraction from protein and carbohydrates and the total energy intake were calculated from a single 24-hour dietary recall.

We limited the analyses to participants 20 years or older who attended the medical examination and included 13907 participants (6825 men and 7082 women) with complete information in our analyses. We treated glycemic index, carbohydrate intake, and glycemic load as either continuous or categorical variables. Means or percentages for HDL-C concentration and baseline characteristics were calculated for quintiles of glycemic index and glycemic load. To examine the significance of means or percentages of these variables by quintiles of glycemic index and glycemic load, we performed tests for linear trend. Least squares–adjusted means of HDL-C concentration were calculated using analysis of covariance. Tests for linear trend were performed using regression analysis by assigning the median values of glycemic index or glycemic load for each of the quintiles. All analyses were done using computer software (version 7.5, Software for the Statistical Analysis of Correlated Data [SUDAAN]; Research Triangle Park, NC) to obtain proper variance estimates because of the complex sampling design.

ucts.[11] Examples of foods with a higher glycemic index include potatoes, white breads with refined flour, and refined grain cereals. Recently, several studies found that the glycemic index is positively associated with the incidence of type 2 diabetes mellitus[12,13] and cardiovascular disease.[14]

Because persons consuming diets with a high glycemic index may have lower HDL-C concentrations than persons consuming a diet with a lower glycemic index, thus indirectly increasing the risk of coronary heart disease, we examined data from the Third National Health and Nutrition Examination Survey (NHANES III) to examine the relationship between glycemic index and HDL-C concentrations in the US population.[15,16]

## RESULTS

For glycemic index, the rounded quintiles were 75% or less, 76% to 79%, 80% to 83%, 84% to 87%, and 88% or higher. For glycemic load, the quintiles were 98 or less, 99 to 127, 128 to 157, 158 to 198, and 199 or higher. As the glycemic index increased, decreasing trends were noted for age, white race, education, and BMI (**Table 1**). The percentage of men, current smokers, and physically inactive participants was directly associated with increasing glycemic index. Except for education, current smoking, and physical inactivity, the relationships between glycemic load and the other variables were similar to those described for glycemic index (**Table 2**).

As glycemic index increased, the unadjusted mean serum HDL-C concentration decreased 8%, from 1.38 to 1.27 mmol/L (**Table 3**). After adjustment for age, sex, race or ethnicity, education, smoking status, BMI, alcohol intake, physical activity, energy fraction from protein and carbohydrates (quintiles), and total energy intake (quintiles), this relationship changed little. Eliminating participants with glucose concentrations of 7 mmol/L or higher or those with pre-

**Table 1. Unadjusted Means and Percentages of Selected Covariates by Quintiles of Glycemic Index***

| | Quintiles, % | | | | | |
|---|---|---|---|---|---|---|
| | ≤75 (n = 2796) | 76-79 (n = 2847) | 80-83 (n = 2915) | 84-87 (n = 2767) | ≥88 (n = 2582) | *P* for Trend |
| Glycemic index | | | | | | |
| Mean | 70.7 | 77.4 | 81.3 | 85.1 | 90.7 | ... |
| Median | 71.7 | 77.5 | 81.3 | 85.0 | 89.9 | ... |
| Age, y | 49.8 (0.7) | 45.4 (0.6) | 43.8 (0.6) | 42.4 (0.7) | 42.4 (0.4) | <.001 |
| Men | 40.7 (1.6) | 46.7 (1.5) | 52.2 (1.3) | 56.0 (0.9) | 53.3 (1.4) | <.001 |
| White race | 82.9 (1.5) | 78.4 (1.6) | 75.2 (1.5) | 74.4 (1.4) | 76.6 (2.2) | <.001 |
| Education, y | 12.8 (0.1) | 12.5 (0.1) | 12.6 (0.1) | 12.1 (0.1) | 12.0 (0.1) | <.001 |
| Current smoker | 19.5 (1.4) | 24.8 (1.5) | 27.4 (1.3) | 32.9 (1.9) | 37.3 (1.6) | <.001 |
| Body mass index, kg/m² | 26.8 (0.1) | 26.8 (0.2) | 26.5 (0.2) | 26.2 (0.1) | 26.3 (0.2) | .002 |
| Inactive | 10.6 (0.9) | 13.2 (1.1) | 12.8 (1.1) | 15.4 (1.2) | 19.3 (1.2) | <.001 |
| Alcohol intake, drinks/wk | 11.6 (1.0) | 12.7 (1.9) | 13.3 (2.4) | 8.0 (0.6) | 10.2 (2.4) | .23 |

**Participants, aged 20 years or older, were from the Third National Health and Nutrition Survey, 1988-1994.[15,16] Data are presented as percentage (SE) unless indicated otherwise. Ellipses indicate not applicable.*

**Table 2. Unadjusted Means and Percentages of Selected Covariates by Quintiles of Glycemic Load***

| | Quintiles | | | | | |
|---|---|---|---|---|---|---|
| | ≤98 (n = 2617) | 99-127 (n = 2554) | 128-157 (n = 2666) | 158-198 (n = 2808) | ≥199 (n = 3262) | *P* for Trend |
| Glycemic load | | | | | | |
| Mean | 76.4 | 112.8 | 141.8 | 176.3 | 261.6 | ... |
| Median | 80.1 | 112.7 | 141.6 | 175.6 | 238.2 | ... |
| Age, y | 46.7 (0.6) | 45.7 (0.5) | 46.0 (0.7) | 44.1 (0.8) | 41.3 (0.6) | <.001 |
| Men | 44.4 (1.4) | 44.6 (1.5) | 50.0 (1.1) | 50.9 (1.7) | 59.0 (1.0) | <.001 |
| White race | 80.2 (1.4) | 81.7 (1.5) | 78.8 (1.8) | 77.6 (1.6) | 69.2 (1.7) | <.001 |
| Education, y | 12.4 (0.1) | 12.5 (0.1) | 12.6 (0.1) | 12.5 (0.1) | 12.0 (0.1) | .02 |
| Current smoker | 31.1 (1.4) | 26.9 (1.6) | 25.6 (1.0) | 26.0 (1.7) | 32.3 (1.7) | .68 |
| Body mass index, kg/m² | 27.6 (0.2) | 26.7 (0.2) | 26.6 (0.2) | 25.9 (0.2) | 25.9 (0.1) | <.001 |
| Inactive | 14.3 (1.2) | 14.4 (1.2) | 14.4 (1.3) | 13.5 (1.1) | 14.6 (1.0) | .94 |
| Alcohol intake, drinks/wk | 11.1 (2.0) | 8.7 (0.5) | 9.9 (0.9) | 11.3 (1.8) | 14.8 (2.5) | .13 |

**See footnote to Table 1. Glycemic load is the product of glycemic index and carbohydrate intake (see "Methods" section).*

viously diagnosed diabetes mellitus did not change our results materially. The multiply adjusted HDL-C concentrations were 1.37, 1.33, 1.32, 1.29, and 1.29 mmol/L for quintiles 1 through 5 of glycemic index, respectively ($P<.001$ for trend). The decrease in HDL-C concentration with increasing glycemic index was more pronounced among men (unadjusted 7% decrease; adjusted 8% decrease) than women (unadjusted 5% decrease; adjusted 2% decrease). The decrease in percentage of HDL-C concentration was similar for participants with a BMI less than 25 and 25 or higher. Adjusting for the same covariates and considering glycemic index as a continuous variable, we found a change in HDL-C concentration of –0.06 mmol/L per 15-unit increase in glycemic index ($P<.001$). The multiple $R^2$ for the model was 0.23.

The pattern of decreases in HDL-C concentration with increases in glycemic load were similar to those described for glycemic index except that the sex differences noted for glycemic index were less pronounced (**Table 4**). The multiple $R^2$ for the model was 0.20. Eliminating participants with glucose concentrations of 7 mmol/L or higher or those with previously diagnosed diabetes mellitus did not change our results materially. The multiply adjusted HDL-C concentrations were 1.36, 1.31, 1.32, 1.31, and 1.29 mmol/L for quintiles 1 through 5 of glycemic load, respectively ($P=.007$ for trend).

## COMMENT

In this representative sample of US adults, we found that a lower quality of carbohydrate intake as characterized by a high glycemic index was associated with a lower HDL-C concentration, independent of factors known to be associated with HDL-C concentrations and the amount of carbohydrate intake. Our results are consistent with findings from a survey conducted by Frost et al[6] in a national sample of adults in England. They recently reported that glycemic index and HDL-C concentration were inversely related among 1420 British participants aged 18 to 64 years in a cross-sectional study. The percentage decrease in HDL-C concentration among men in their study was similar to that observed in our study. They found a stronger inverse relationship between glycemic index and HDL-C concentration among women than men, however. These differences are likely due to differences in the demographic compositions of the 2 samples and to the use of different dietary instruments.

Because our results were derived from a cross-sectional study, we cannot conclude that changes in the glycemic index and glycemic load can cause changes in HDL-C concentrations. The food frequency questionnaire that we used was an abbreviated one and did not

Table 3. Mean Concentrations of High-Density Lipoprotein Cholesterol (HDL-C) According to Quintiles of Glycemic Index*

| HDL-C Concentration | Quintiles, % | | | | | P for Trend |
|---|---|---|---|---|---|---|
| | ≤75 | 76-79 | 80-83 | 84-87 | ≥88 | |
| Participants, No. | 2796 | 2847 | 2915 | 2767 | 2582 | ... |
| Crude | 1.38 (0.01) | 1.32 (0.01) | 1.29 (0.01) | 1.26 (0.01) | 1.27 (0.01) | <.001 |
| Age adjusted† | 1.38 (0.01) | 1.32 (0.01) | 1.30 (0.01) | 1.26 (0.01) | 1.27 (0.01) | <.001 |
| Age and BMI adjusted† | 1.39 (0.01) | 1.32 (0.01) | 1.30 (0.01) | 1.25 (0.01) | 1.27 (0.01) | <.001 |
| Multivariate†‡ | 1.36 (0.01) | 1.31 (0.01) | 1.30 (0.01) | 1.27 (0.01) | 1.28 (0.01) | <.001 |
| Men, No. | 1226 | 1355 | 1478 | 1444 | 1322 | ... |
| Crude | 1.23 (0.02) | 1.20 (0.01) | 1.20 (0.01) | 1.15 (0.02) | 1.14 (0.02) | <.001 |
| Age adjusted† | 1.23 (0.02) | 1.20 (0.01) | 1.20 (0.01) | 1.15 (0.02) | 1.14 (0.02) | <.001 |
| Age and BMI adjusted† | 1.24 (0.02) | 1.21 (0.01) | 1.20 (0.01) | 1.14 (0.02) | 1.14 (0.02) | <.001 |
| Multivariate†§ | 1.24 (0.02) | 1.21 (0.01) | 1.20 (0.01) | 1.14 (0.02) | 1.14 (0.02) | <.001 |
| Women, No. | 1570 | 1492 | 1437 | 1323 | 1260 | ... |
| Crude | 1.49 (0.02) | 1.42 (0.02) | 1.40 (0.02) | 1.39 (0.02) | 1.42 (0.02) | .001 |
| Age adjusted† | 1.49 (0.02) | 1.42 (0.02) | 1.40 (0.02) | 1.40 (0.02) | 1.42 (0.02) | .009 |
| Age and BMI adjusted† | 1.49 (0.02) | 1.42 (0.02) | 1.40 (0.02) | 1.39 (0.02) | 1.42 (0.02) | .001 |
| Multivariate†§ | 1.47 (0.01) | 1.42 (0.02) | 1.40 (0.02) | 1.40 (0.01) | 1.44 (0.02) | .06 |
| Participants with BMI <25, No. | 991 | 1103 | 1158 | 1149 | 1117 | ... |
| Crude | 1.52 (0.02) | 1.46 (0.02) | 1.42 (0.02) | 1.38 (0.02) | 1.38 (0.02) | <.001 |
| Age adjusted | 1.51 (0.02) | 1.45 (0.02) | 1.42 (0.02) | 1.38 (0.02) | 1.39 (0.02) | <.001 |
| Age and BMI adjusted | 1.52 (0.02) | 1.46 (0.02) | 1.42 (0.02) | 1.37 (0.02) | 1.38 (0.02) | <.001 |
| Multivariate‡ | 1.48 (0.02) | 1.44 (0.02) | 1.43 (0.02) | 1.39 (0.02) | 1.41 (0.02) | .003 |
| Participants with BMI ≥25, No. | 1805 | 1744 | 1757 | 1618 | 1465 | ... |
| Crude | 1.28 (0.01) | 1.22 (0.01) | 1.19 (0.01) | 1.16 (0.01) | 1.17 (0.02) | <.001 |
| Age adjusted | 1.27 (0.01) | 1.22 (0.01) | 1.20 (0.01) | 1.16 (0.01) | 1.17 (0.02) | <.001 |
| Age and BMI adjusted | 1.27 (0.01) | 1.22 (0.01) | 1.20 (0.01) | 1.16 (0.01) | 1.17 (0.02) | <.001 |
| Multivariate‡ | 1.26 (0.01) | 1.22 (0.01) | 1.20 (0.01) | 1.18 (0.01) | 1.18 (0.01) | <.001 |

*Participants, aged 20 years and older, were from the Third National Health and Nutrition Survey, 1988-1994.[15,16] Data are presented as millimoles per liter unless indicated otherwise; to convert millimoles per liter to milligrams per liter, divide by 0.0259. BMI indicates body mass index, which is calculated as weight in kilograms divided by the square of height in meters; ellipses, not applicable.

†Adjusted using analysis of covariance.

‡Adjusted for age, sex, race or ethnicity, education, smoking status, BMI, alcohol intake, physical activity, energy fraction from protein (quintiles) and carbohydrates (quintiles), and total energy intake (quintiles).

§Adjusted for age, race or ethnicity, education, smoking status, BMI, alcohol intake, physical activity, energy fraction from protein (quintiles) and carbohydrates (quintiles), and total energy intake (quintiles).

include questions about portion size, which inevitably led to some misclassification of participants' dietary intake. Consequently, we probably underestimated the magnitude of the decreases in HDL-C concentration with increases in glycemic index or glycemic load. We may have failed to account for relevant confounders or incompletely adjusted for selected confounders. Finally, the apparent association between glycemic index or glycemic load and HDL-C concentration may be due to another nutrient.

Although it has long been known that diet can affect circulating concentrations of HDL-C, much remains to be learned about how specific aspects of diet can affect HDL-C concentrations. Thus, the recent research that showed that the quality of the dietary carbohydrate composition can affect HDL-C concentrations independent of the quantity of carbohydrate intake suggests a new pathway through which a diet with a low glycemic index may lower the risk of coronary heart disease. A summary of observational studies[3] found that for every decrease of about 0.026 mmol/L in HDL-C concentration, the risk of coronary heart disease increased by 1.9% to 2.3% in men and 3.2% in women. Thus, an increase in the adjusted HDL-C concentration of about 8% in men and 2% in women could theoretically reduce coronary heart disease by about 8% in men and 3.5% in women. In the United States in 1996, about 242 036 men and 234 088 women died of coronary heart disease, and about 1.1 million Americans were estimated to have had myocardial infarction or fatal coronary heart disease.[23] Therefore, the potential reductions in coronary heart disease events and mortality due to changes in HDL-C concentrations, such as those we found, are not trivial.

Although metabolic studies in patients with diabetes mellitus or hyperlipidemia have shown lower total cholesterol concentrations when carbohydrates with a low glycemic index are substituted for those with a high glycemic index, findings on HDL-C concentrations have been less consistent.[24-28] Therefore, the recent findings in 2 national surveys in the United States in the present study and England[6] need to be confirmed by prospective studies or clinical metabolic trials of healthy persons. Because of the long-standing controversy about the potential etiologic role of glycemic index in the pathogenesis of several chronic diseases and the short-term nature of metabolic data, data from large prospective cohorts that directly relate dietary carbohydrates, glycemic index, and glycemic load to the subsequent development of type 2 diabetes mellitus and coronary heart disease will be most informative. In the final analysis, the ultimate criterion by which to judge the clinical utility of the glycemic index or that of any other classification schemes of foods is its ability to predict disease outcomes. If dietary carbohydrate composition influences HDL-C concentrations, recommendations about the best dietary sources of carbohydrates may need to be refined. In light of recent data suggesting that dietary carbohydrate composition may affect the risk of type 2 diabetes

**Table 4. Mean Concentrations of High-Density Lipoprotein Cholesterol (HDL-C) According to Quintiles of Glycemic Load***

| HDL-C Concentration | Quintiles | | | | | *P* for Trend |
|---|---|---|---|---|---|---|
| | ≤98 | 99-127 | 128-157 | 158-198 | ≥199 | |
| Participants, No. | 2796 | 2847 | 2915 | 2767 | 2582 | ... |
| Crude | 1.38 (0.01) | 1.32 (0.01) | 1.29 (0.01) | 1.26 (0.01) | 1.27 (0.01) | <.001 |
| Age adjusted† | 1.34 (0.01) | 1.32 (0.02) | 1.31 (0.01) | 1.31 (0.01) | 1.26 (0.01) | <.001 |
| Age and BMI adjusted† | 1.36 (0.01) | 1.32 (0.02) | 1.31 (0.01) | 1.29 (0.01) | 1.24 (0.01) | <.001 |
| Multivariate†‡ | 1.35 (0.01) | 1.31 (0.01) | 1.31 (0.01) | 1.30 (0.01) | 1.26 (0.01) | <.001 |
| Men, No. | 1166 | 1151 | 1320 | 1393 | 1795 | ... |
| Crude | 1.21 (0.02) | 1.19 (0.02) | 1.20 (0.02) | 1.16 (0.02) | 1.17 (0.01) | .05 |
| Age adjusted† | 1.21 (0.02) | 1.19 (0.02) | 1.20 (0.02) | 1.16 (0.02) | 1.16 (0.01) | .03 |
| Age and BMI adjusted† | 1.24 (0.02) | 1.19 (0.02) | 1.19 (0.02) | 1.15 (0.01) | 1.15 (0.01) | <.001 |
| Multivariate†§ | 1.24 (0.02) | 1.20 (0.02) | 1.19 (0.01) | 1.15 (0.01) | 1.14 (0.01) | .001 |
| Women, No. | 1451 | 1403 | 1346 | 1415 | 1467 | ... |
| Crude | 1.45 (0.02) | 1.43 (0.02) | 1.42 (0.02) | 1.46 (0.02) | 1.38 (0.02) | .04 |
| Age adjusted† | 1.44 (0.02) | 1.43 (0.02) | 1.42 (0.02) | 1.46 (0.02) | 1.38 (0.02) | .05 |
| Age and BMI adjusted† | 1.46 (0.02) | 1.43 (0.02) | 1.42 (0.02) | 1.44 (0.02) | 1.37 (0.02) | <.001 |
| Multivariate†§ | 1.46 (0.02) | 1.42 (0.02) | 1.42 (0.01) | 1.44 (0.02) | 1.38 (0.02) | .002 |
| Participants with BMI <25, No. | 871 | 945 | 1046 | 1215 | 1441 | ... |
| Crude | 1.49 (0.02) | 1.48 (0.03) | 1.43 (0.02) | 1.41 (0.02) | 1.36 (0.02) | <.001 |
| Age adjusted | 1.49 (0.02) | 1.48 (0.03) | 1.43 (0.02) | 1.41 (0.02) | 1.36 (0.02) | <.001 |
| Age and BMI adjusted | 1.49 (0.02) | 1.48 (0.03) | 1.43 (0.02) | 1.41 (0.02) | 1.36 (0.02) | <.001 |
| Multivariate‡ | 1.48 (0.02) | 1.46 (0.03) | 1.43 (0.02) | 1.41 (0.02) | 1.38 (0.02) | <.001 |
| Participants with BMI ≥25, No. | 1746 | 1609 | 1620 | 1593 | 1821 | ... |
| Crude | 1.26 (0.02) | 1.20 (0.02) | 1.21 (0.01) | 1.20 (0.01) | 1.15 (0.01) | <.001 |
| Age adjusted | 1.25 (0.02) | 1.20 (0.02) | 1.21 (0.01) | 1.20 (0.01) | 1.16 (0.01) | <.001 |
| Age and BMI adjusted | 1.26 (0.02) | 1.20 (0.02) | 1.21 (0.01) | 1.20 (0.01) | 1.15 (0.01) | <.001 |
| Multivariate‡ | 1.26 (0.01) | 1.20 (0.01) | 1.21 (0.01) | 1.20 (0.01) | 1.16 (0.01) | <.001 |

**See footnotes to Table 3. In the last 2 footnotes, HDL-C concentrations were adjusted for energy fraction from fat, rather than from carbohydrates. Glycemic load is the product of glycemic index and carbohydrate index (see "Methods" section).*

mellitus and coronary heart disease, it may be prudent to consider substituting foods with a low glycemic index for ones with a high glycemic index as a means of lowering the risk of developing these conditions.

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